If there is no threshold effect and the dose/response relationship goes to zero risk for zero exposure, which is still possible with present data, then the extremely small assumed risks at 1 µW/sq.cm, when applied to a very large population, could become statistically significant.

Support for this view would be available if large populations of workers or residents who had been exposed to above average intensities of microwaves could be identified and studied, with some knowledge of the level of microwave exposure.

The absence of specifically directed studies involving a comprehensive assessment of potential health effects probable leads to a major under-estimate of the possible adverse effects. This arises because mortality statistics are more robust and more readily available than is the incidence of non-fatal tumors and lesions for example. Demers et.al. demonstrate this using a Tumor Registry verses Death Certificates in an Occupational Cohort Studies in the United States (Am. Jour. of Epidem.;136,10: 1232-1240). Their abstract records the following conclusion:

"As expected, an increased ability to study relatively common cancers with low fatality rates was demonstrated by the incidence data. The most dramatic example was seen for bladder cancer. Twenty-four bladder cancers were diagnosed among the study cohort (consisting of 4,528 Tacoma fire fighters and police officers) between 1974 and 1989, whereas only two deaths were attributed to this malignancy."

Hence studies of the full potential impact of any particular environmental stressor are difficult and rare because of the limitations in available data and the complexity of human subjects. This is more likely to lead to an under-estimate of the impact of a particular stressor than an over-estimate.

#### 8.2 Physical Therapists and Miscarriage:

A study of American Physical Therapists (American Journal of Epidemiology, Vol 138, No10, November 15 1993) investigated the incidence of miscarriage in a sample of over 19,000 female therapists. A striking result of this study shows that for miscarriages occurring before the 7th week of gestation, of those using microwave diathermy, 47.7 % miscarried compared to 14.5 % for the control group, Figure 3. No such difference was found for practices using shortwave diathermy. Shortwave diathermy uses 27.12 MHz (11 m wavelength) while microwave diathermy uses 915 MHz (32.8 cm wavelength) and 2,450 MHz (12 cm wavelength).

Table 1: Unconditional odds ratios for the association between risk of recognised miscarriage and reported exposure to microwave diathermy during the 6 months prior to and the first trimester of pregnancy: Physical Therapists Study, 1989-1990

	No. of exposures	Case pregnancies	Control pregnancies	OR*(95% Ci)	× <sup>2</sup> test for trend
All pregnancies	0 <5 5-20 >20 Total no. expe	1,459 88 72 45 osed 209	1,494 86 49 29 167	1.00 1.05 (0.77-1.43) 1.50 (1.04-2.17) 1.59 (0.99-2.55) 1.28 (1.02-1.59)	p>0.005
No prior fetal loss	0 <5 5-20 > 20 Total no. expo	1,102 71 58 34 osed 167	1,258 76 47 25 151	1.00 1.07 (0.78-1.49) 1.41 (0.95-2.09) 1.55 (0.92-2.61) 1.26 (1.00-1.59)	p>0.01

The odds ratios for all pregnancies is highly significant ( $\rho$ <0.005). Similar figures were obtained for those who reported no previous miscarriage, but with a smaller sample the significance is p>0.01.

Measurements of the leakage from the microwave equipment at 15 cm from the source was as high as 15,000  $\mu$ W/cm² but exposure at waist level ranges from 80 to 1,200 $\mu$ W/cm². The therapist only spends a few minutes setting up the equipment, 0.5 to 1 minute near the machine while it is on, and then she leaves for the remaining treatment time which is typically 20 minutes. A significant occupational exposure will only occur then if many treatments are given and the operator stands very close to the equipment for prolonged periods. Assuming a conservatively long estimate of 2 minutes exposure per treatment, the per treatment dosage is 0.0096 to 0.144 J/cm².

Treatment rates ranged from <5 to >20 per month. One treatment per month averages 0.004 to  $0.056\mu\text{W/cm}^2$ , 10 per month 0.04 to  $0.56\mu\text{W/cm}^2$  and 20 treatments per month 0.08 to  $1.11\mu\text{W/cm}^2$ . The lowest limit is very difficult to estimate with reliability but the mean level of the middle band is close to  $0.3\mu\text{W/cm}^2$ .

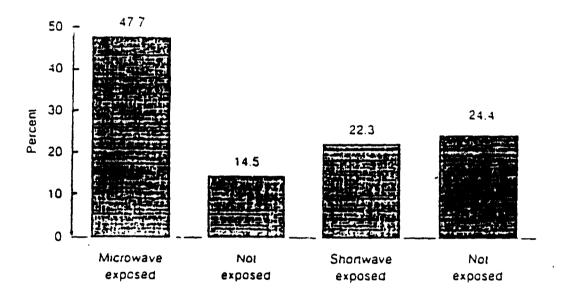


Figure 3: Proportion of miscarriages occurring before the seventh week of gestation, by exposure status: Physical Therapists Study 1989-90, Ouellet-Hellstrom and Stewart (1993).

This suggests that a two- to three-fold increase in miscarriage occurred with a mean monthly microwave exposure of somewhat less than  $1\mu W/cm^2$ . It is possible that exposures need to be in the range of 80 to  $1200\mu W/cm^2$  to cause any effect through in vitro absorption by the foetus in the womb. It still remains possible, because of the increasing dose-response relationship, that cumulative damage could also be occurring because of cellular changes which could conceivably accumulate with continuous exposure to mean intensities of 0.025 to  $0.6\mu W/cm^2$  (choosing the means from 1 to 20 treatments per month).

The authors summarize their findings as follows:

"In summary, in this study, women who reported using microwave diathermy at the time of conception were at increased risk of miscarriage. The risk increased with increasing exposure, and persisted even when known confounders were taken into consideration in the analysis. Whether the excess risk is associated directly with the use of microwave diathermy per se or with something closely related to its use has yet to be determined. Women who reported using shortwave diathermy were not at increased risk."

The results of this survey are sufficiently significant, applying the precautionary principle, to require adoption of a spatial separation between a cellsite and schools or residences where pregnant women might be. Choosing the mean of the level where the first significant increase in miscarriage is observed (5-20, averaging 10), 0.04 to  $0.56\mu\text{M/cm}^2$ , applying a safety factor of 10 to the highest level brings it close to the lowest level and suggests a safety limit of about  $0.05\mu\text{M/cm}^2$ . For a typical TELECOM site this will be over 500 m.

#### 8.3 Testicular Cancer and self-reported microwave exposure:

A paper by Richard B. Haynes et.al. ("Occupation and Risk for Testicular Cancer: A Case-Control Study, International Journal of Epidemiology, vol 19, No.4, pp 825-831, 1990) reports the following data and comments:

Table 2: Odds ratio (OR)\* for testicular cancer by self-reported exposure.

Exposure	No. of Controls	All Cancer (N = 271)		
		OR	(95% Cl <sup>-</sup> )	
Radioactive Material	23	1.2	0.6-2.3	
Radar equipment	38	1.1	0.7-1.9	
Microwave and other radiowaves	10	3.1	1.4-6.9	
Hydrocarbons	12	1.5	0.7-3.4	
Pesticides	29	1.2	0.4-1.2	

Notes: \* Odds ratio, adjusted for age (16-21, 22-25, 26-29, 30-45)

+ 95% CI = 95% confidence interval

The authors report that "When enquiries were made about specific exposures, a statistically significant association was found for reported exposure to microwaves and other radiowaves. The finding held for both seminomas and other tumor types. However an independent assessment by job title did not support this finding." .... "The assessment of the potential association, if any, of low-frequency electromagnetic radiation and cancer risk is hampered by uncertainties about selective electromagnetic frequency ranges, the clear lack of a clear biological mechanism, as well as by difficulties of exposure assessment."

This summarises the Epidemiologists' dilemma about microwave risk in populations. The current lack of a known biological mechanism and the lack of monitoring of actual exposures, over a spectrum of frequencies, limits the researcher's ability to come to firm conclusions, even in the face of case after case of statistically significant results.

I submit that the range and strength of the evidence, taken together, is sufficient to act to minimise public exposure to electromagnetic radiation, especially microwaves, because of their resonance with objects of the range of sizes of human bodies and body parts, the increased number of early pregnancy miscarriages and testicular cancer.

#### 8.4 The Goldsmith Review:

A recently published review article, Goldsmith (1995), summarises some evidence which does show potential and actual health effects in humans from microwave exposure at levels which are generally somewhat below the New Zealand standard NZS6609. Dr Goldsmith reviews data and findings which have been published in peer-reviewed international journals as well as his own research into the effects of low level microwave exposure on the staff of the United States embassy in Moscow. At a recent meeting in Chartwell School Dr David Black, Occupational Health Consultant, attempted to dismiss and discredit this paper. Dr Black claimed that it was only published with reluctance and under pressure. His statement strongly implied that Dr Goldsmith is not a credible nor reliable scientist and doctor. I strongly challenge this.

Dr John R. Goldsmith, Professor of Epidemiology, faculty of Health Sciences, Ben-Gurion University of Negev, Israel, is an eminent and internationally recognised and respected epidemiologist who has told me personally that he had no trouble at all with his reviewers nor the editor in getting this paper published.

I have studied several of Dr Goldsmith's references which are published in journals which are held by the Medical School Library in Christchurch and Dr Goldsmith's paper fairly represents the findings of these papers.

For example, the study of miscarriages among female physiotherapists in the United States.

Dr Goldsmith carried out his own review of all published documents surrounding the U.S. Moscow embassy exposure, including significant material obtained under the Official Information Act and not previously published. Dr Goldsmith points out, as other researchers have concluded also, that concentration on death as an outcome, ignores a host of evidence of changed health status which is statistically correlated to increased exposure to microwaves.

It was found that despite the lower than average age of the embassy staff and families than the general U.S. population, there was a statistically significant increase with white cell counts, mean hematocrit increased and a threefold increase in monocyte count, while neutrophil percentage fell and then rose and the reverse occurred for lymphocytes. There were raised cancer deaths (15 out of 31 women staff), including leukemia, female genitalia cancer and child cancer.

Adult dependents showed five malignant neoplasms compared with 1.5 expected and dependent children showed greater than expected anemia.

In the Moscow embassy case the mean exposure levels are estimated at between  $5\mu W/cm^2$  and  $18\mu W/cm^2$ , compared to the "safe level" of the New Zealand standard of 200  $\mu W/cm^2$ . Assuming an average 40 hour working week, the mean exposure of embassy staff is in the range 1  $\mu W/cm^2$  to 4  $\mu W/cm^2$ .

In his discussion Dr Goldsmith makes the following observation which is significant from a person of his standing, but is shared by every researcher of the five I have contacted so far:

There are strong political and economic reasons for wanting there to be no health effect of RF/MW (radiofrequency/microwave) exposure, just as there are strong public health reasons for more accurately portraying the risks. Those of us who intend to speak for public health must be ready for opposition that is nominally but not truly, scientific."

#### 8.5 Chinese research:

Totally separate and independent of Dr Goldsmith's work there are several other researchers and research groups who have detected significant changes in health status in workers exposed to microwaves. The following abstract appears among those from the Second Annual Meeting of the International Society for Environmental Epidemiology, Berkeley, California, August 13-15, 1990. Judging by the terminology and the author's name this is presumed at this stage to be based on work in China. Work is going on in China which has been reported in Microwave News.

I quote the abstract in full:

#### "EFFECT OF MICROWAVE RADIATION ON HUMAN HEALTH

W Shao-Guang, et.al.

The effects of microwave radiation (i.e. continued wave and pulse wave) in two exposed groups (TEGs) were investigated. Results showed that the neurasthenic syndrome appeared in 38.3 % of the continued wave group, 40.2% of the pulse wave group and 5.3 % of the control group. Cases showing autonomic nerve dysfunction in the cardiovascular system and opacity at the posterior capsule of the lens occurred more frequently in the TEG than in the control group. Platelet, cholinesterese activity, and serum IgG and IgM levels were below normal in the TEG compared with the control group (p<0.05). But no obvious change in leukocytes and alkaline phosphatase was found.

It is therefore concluded that workers exposed to pulsed waves are at higher risk than those exposed to continued waves."

In the face of it these are very significant results. However, without checking the original paper in full and checking with the authors this should remain as a potential example only. I am interested to find out why the increase of 33 to 35 % above the control group for those exposed to microwaves is not commented on in the abstract.

#### 8.6 Suicide among electric utility workers in England and Wales

The psychological stresses reported in Chinese workers exposed to microwaves is reflected in some inconclusive studies in England and Wales (British Joun. of Indust. Med. 1990;47:788-789). An earlier study (1970-72) showed a significant increase in proportional mortality ratios (PMR) for suicide among:

Table 3: Suicide mortality for men aged 15-64 in electrical occupations: England and Wales 1970-72.

Occupational Group	Observed	Expected	PMR	95% CI
Telegraph radio operators	10	3.9	256	(123-471)
Electronic engineers (professional)	16	10.2	156	(89-253)
Radio and radar mechanics	19	12.4	153	(92-239)
Total (all groups not just these)	154	173	89	(75-104)

Overall electrical workers appear to have a lower than average incidence of suicide in England and Wales but the three groups highlighted have a higher than average incidence of suicide. These are groups whose work involves the radiation of EM shortwave and microwave radiation, whereas the other groups cited, such as Installers and repairment (telephones), electricians, electrical and electronic fitters, etc are much more involved with mains electricity.

In the follow up study (1979-80 and 1982-83) it was found that electrical workers overall were at slightly higher than average risk of suicide PMR = 102 (CI:91-113), however the occupational groups had been reorganised into different categories which failed to separate those who were involve primarily with mains electricity and those involving radiated microwaves and shortwave radio signals. Telephone operators were grouped with radio/telegraph operators for example. This effectively masked any relationship which may exist for groups working specifically with microwaves, as in the case of the Chinese workers.

This makes the earlier \*970-72 study more relevant. The concern remains then about the mental stress identified by the Chinese and is potential implications for increased suicide.

#### 8.7 German Brain Function Research: EEG and microwave exposure

I have been able to locate and correspond with Dr Lebrecht von Klitzing, Medical Physicist at the Medical University of Luebeck (Germany (West)). I was able to verify that he has demonstrated and measured changes in the EEG traces of student's brains when exposed to very low levels of pulse microwave radiation using the magnetic part of the wave. The intensity levels used were less than  $1\mu W/cm^2$  in the student's brain. I have asked him to estimate or measure the intensity level outside the student's heads to obtain an ambient exposure level. He used a 150 MHz carrier and a 217 Hz modulation.

I have further asked Dr von Klitzing to comment on extension of his results to 800 MHz and 900 MHz, and if possible carry out an experiment using 800 to 900 MHz carriers, the frequency of our cellphone networks in New Zealand.

In a German magazine article (Wohnung + Gesundheit) Dr von Klitzing points out that modern digital cellphone networks work on pulses at about 217 Hz. He says "High frequency fields pulsed at low frequency with very low power have an effect on the human EEG. Is the intercellular communication system disturbed by these fields? It is possible. The explanation is unknown at this stage. Nevertheless there are biological effects."

The exposure levels which Dr von Klitzing is using in his laboratory experiments is similar to those used every day 100 to 500 m from a 50 Watt cellsite transmitter.

During a digital cellphone demonstration in Erkrath, near Dusseldorf on 14th March 1994, Dr von Klitzing said "Biological effects appear with  $0.1\mu W/cm^2$  field strength. When I expose a person to a typical 217 Hz signal of the digital cellphone transmission then the EEG shows a high peak within the 10 Hz area. The EEG reacts only with constantly repeated pulsing and does not react when it is variable. The EEG shows peaks and curves under the influence of digital transmissions which has not been seen before. Peaks that were not recognizable. Amazingly these peaks persist a long time after the exposure stops... from a few hours up to days and even up to a week."

I faxed the English translation of the German article to Dr von Klitzing which he verified was correct except for accuracies sake he requested that a sentence be removed, which I have done. I have also checked Dr von Klitzing's reputation as a researcher with Dr Bruce Spittle, Otago Medical School, Senior Lecturer in Psychological Medicine.

In a letter dated 9 March 1995, Dr Spittle says "I have found his (Dr von Klitzing) work to be sound and thoroughly done. His work supports studies by Ross Adey, e.g. Bioelectromagnetics 1982; 3:295-307 that low frequency modulation of a high frequency carrier wave may produce biological effects. My interpretation of the von Klitzing work is that pulsed digital repeaters may have a more profound effect that pulsed am repeaters."

I have been discussing possible hypotheses of the physical processes by which the pulsed signal may enter the biological/electromagnetic structure of the brain. The physical hypothesis we are debating and testing is that the microwave carries the energy into the brain tissue and that the modulation frequency resonates with the alpha and beta rhythms of the brain. The preliminary results presented in the latest von Klitzing paper support this hypothesis, see Figure 4 for example.

In a fax dated 7 March 1995, Dr von Klitzing replied to my suggestions:

"I think you are on the right way (because this is my way, too) in interpretation (of) the subcellular influences by GSM-standard-telecommunication. Not only EEG is altered, we have shifts in the answer of immunosystems (human lymphozytes) or in the metabolic regulation of cell cultures (yeast cells; these cells have the same metabolic pathway like liver cells, only with lactate instead of ethanol as anaerobic production.

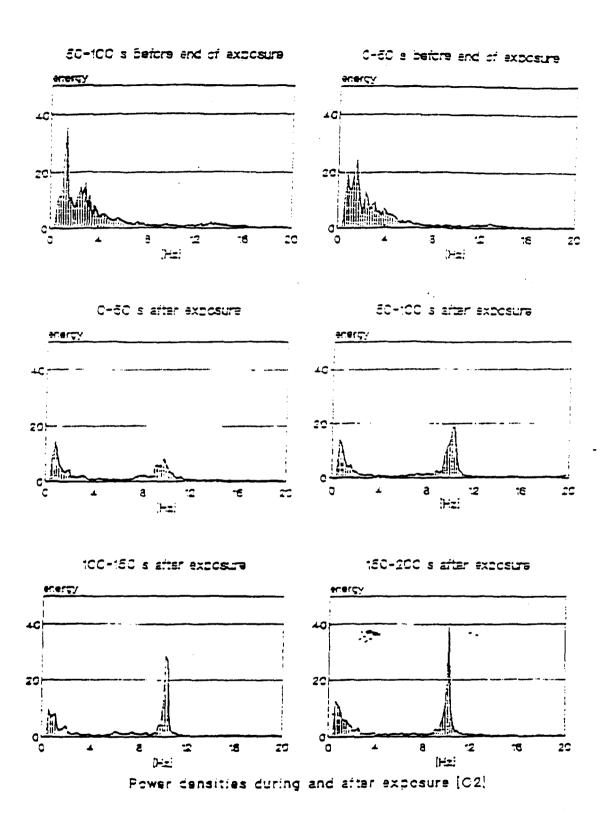


Figure 4: Alpha-EEG (O2-position) of man is altered by pulsed microwave fields, shown for periods during exposure and immediately after exposure to demonstrate prompt energy increase, von Klitzing (1995)

Our hypothesis is that in paths of intercellular communication the periodicity of LF-pulsed HF-signals interfered with the biological "Zeitgeber".

Because of the fact that static magnetic fields alter the spectral density of EEG not only during exposure but for some hours after it, this points to an 'interface' not to be explained by current induction."

I contend that I have soundly negated the hypothesis that there is no evidence of actual and potential human health effects below the New Zealand Standard of 200  $\mu$ W/cm². There is strong, published, peer-reviewed evidence of severe adverse health effects at mean occupational exposure to microwaves at levels of less than  $0.1\mu$ W/cm². This comes from both brain research and the epidemiological studies.

#### 8.8 New Zealand Epidemiological Study - Electrical Workers

Pearce et.al. (1989) report a case-control study of cancer incidence in electrical workers in New Zealand since previous studies had already suggested that electrical workers were at higher risk of Leukemia. The study, involving 19,904 males on the New Zealand Cancer Registry, who were aged over 20 years at the time of registration. The study concluded that "Firstly, there is an elevated leukaemia risk for New Zealand electrical workers (OR = 1.62; CI = 1.04 - 2.52), but little evidence of increased risks for other cancer sites. Secondly, contrary to other published studies, the increased risk was primarily for chronic leukaemia (OR=2.12) rather than acute leukaemia (OR=1.25), and for lymphatic leukaemia (OR=1.73) rather than myeloid leukaemia (OR=1.22). Third, the increased risk was strongest for certain categories of electrical work including radio and television repairers (OR=7.86; CI=2.20-28.09), electricians (OR=1.68; CI=0.75-3.79), linemen (OR=2.35; CI=0.97-5.70) and power station operators (OR=3.89; CI=1.00-15.22)."

Because of the small sample size the authors warn that the findings "should also be regarded with due caution" but they also note that "These findings are generally consistent with the electromagnetic field hypothesis, but interpretation is difficult in the current absence of quantification of 'typical' exposures to electromagnetic fields experienced in these types of electrical work."

In Environmental Epidemiology a single study is not necessarily significant, but in combination with many other studies, patterns can be identified, trends observed and findings strengthened.

The results in this study are consistent with higher risk for radiative radio frequency exposure (radio and TV repairers) and those working with active, high voltage or frequent "live exposure" fields (electricians, linemen and power station operators).

Once again the urgent need for exposure monitoring is identified as a major impedement to sound identification of dose/risk relationships and cause and effect identification.

#### 8.9 DNA breakdown and microwave absorption

In figure 1 it is shown that microwaves have a molecular resonance response such as the change in the orientation of asymmetrical molecules. Lerner (1984) records a panel discussion of researchers involved with studies about the biological response to EM radiation:

"If heating does not cause these effects (changes in rate of growth of yeast cells), what does? One hypothesis raised by Dr Swicord is that genetic material resonantly absorbs radiation - that is, such material has natural periods of oscillation close to the frequencies of the imposed EM fields.

Dr Swicord presented evidence of molecular-resonance absorption of energy by genetic material, in particular, purified DNA from E. coli bacteria. He measured DNA absorptions at 11 GHz, demonstrating that DNA could absorb 400 times as much energy as water. There's a very strong interaction of DNA with microwave radiation,' Dr Swicord observed. Equally interesting, he reported, the absorption coefficient for DNA increased as the DNA was broken into smaller and smaller lengths by an enzyme. Such enhanced absorption, he said, means that the nuclei of cells are receiving far more absorbed energy per unit mass than the organism as a whole. For example, an absorption rate of 0.05 mW/g for the organism would yield an absorption rate, at least for some DNA, of 20 mW/g.

Some have questioned how absorption of microwave radiation by DNA could affect its behaviour in genes, since the energies involved are small compared with the thermal energy already present in the molecules. Dr Swicord commented on these views following the panel discussion. 'You have to realise that in resonant absorption of coherent radiation, energy can be absorbed over time, not just with each photon, just as abridge can be destroyed by resonant interaction with soldiers matching in lockstep - each push is small, but the cumulative impact is large,' Dr Swicord told Spectrum. 'Similarly, a DNA molecule can absorb the energy of not just one but many photons. This energy may still be small compared with the total thermal energy, but more than enough to change its function, especially if its all in one oscillative mode and if this excitation is maintained over time.' "

After reading about this 1984 panel discussion and the molecular resonance mechanisms which were demonstrated that microwaves break DNA, I was interested to see the leading headline in Microwave News for November/December 1994:

#### "Microwaves Break DNA in Brain; Cellular Phone Industry Skeptical"

#### The report says:

\*Low-level microwave radiation can cause DNA breaks in the brains of experimental animals, according to studies carried out in the U.S. and India. These new results, which have attracted a lot of interest within the cellular phone industry, suggest that microwaves could act as a cancer-causing agent.

Drs Henry Lai and Narendra Singh of the University of Washington, Seattle, have found that a single two-hour exposure to 2.45 GHz radiation at levels which are currently believed to be safe can increase the number of single-strand in the DNA of the brain cells of rats. 'DNA damage is related to the initiation of cancer' - if there is an error in the repair process, it could lead to a problem. We have a long way to go before we can reach any definitive conclusions,' Dr Lai added, pointing out that, 'DNA breaks may stimulate DNA repair mechanisms, which could lead to a beneficial effect.'

A second animal study, by a group led by Dr Soma Sarkar of the Institute of Nuclear Medicine and Applied Sciences in New Delhi, India, found that DNA in the brains and testes of mice had undergone 'rearrangement' following microwave exposure at the same frequency and approximately the same intensity as in the Lai-Singh study. Sarkar and co-workers concluded that a re-evaluation of the mutagenic potential of microwave radiation 'seems imperative'."

Both of these studies are being or have been published in peer-reviewed scientific journals, the Lai-Singh study in Bioelectromagnetics and the Sarkar et.al study in Mutation Research.

While the 1984 results were investigated by several groups and not replicated, NRPB (1993). Now in 1994/95, with much more sophisicated analysis techniques, the effect has been found again.

Here is clear evidence and probable mechanisms for testicular cancer in men and possible mechanisms for Leukemia, genital cancer and the other cancers reported in electrical workers and in the Moscow U.S. embassy study.

Dr Lai used two different exposure rates and concluded that "there appeared to be a dose-response relationship".

#### 9. HYPOTHESIS 4: Threshold for dose/response relationship?

Hypothesis 4: "There is no threshold for the dose/response relationship for microwave exposure for human beings."

#### 9.1 Other phenomena:

It is the experience of many epidemiological studies that as techniques have improved and analytic instrument precision has increased, many thresholds for chemical and radiation exposure have been pushed progressively lower, suggesting that there is in fact no threshold in the dose/response relationship but that a linear or similar relationship is likely to exist.

There is no suggested threshold for UVB exposure and skin cancer for example. A typical mid-summer "burn time" is about 12 to 20 minutes. Assuming a 30 minute actual exposure, ten life time "burns", totalling 5 hours, at 1 W/m² gives a UVB dosage of 18 kJ/m². This is likely to significantly increase the probability of skin damage and melanoma compared to a "never burnt" person. The probability of skin damage is also related to skin type. Hence the slope of the response/dose curve is different for different people. Some people are much more sensitive than others.

#### 9.2 Microwave studies:

Servantie (1989) states "Almost all of these (standards setting studies) are based on the assumption that there is a linear relationship between the amount of energy carried by radiation and the potential for biological or hazardous effects." Dr Lai reports the likelihood of a dose-response relationship for the breakdown of DNA.

The United States Moscow embassy staff were irradiated by microwaves from November 1962 to August 1963. Assuming a mean intensity of  $10\mu W/cm^2$  over a 40 hour mean working week, then the total dosage is around 624 kJ/m<sup>2</sup>. This is about 35 times higher than the UVB exposure assumed above, but the incidence of fatal cancer is a least 50 times higher in the case of the embassy population compared to the New Zealand population and its incidence of skin cancer death at 0.1/1000 per year.

The strong similarities between the established linear dose/response relationship for ionising radiation, with progressively stricter exposure standards, the evolving appreciation of the similar relationship with UVB radiation, and the probable dose and response/risk relationship for microwave radiation (as for the pregnant physiotherapists and miscarriage), are strongly suggestive of a zero threshold for microwave radiation.

If this is true, then the very low level microwaves intensities from a cellsite will accumulate over time to become equal and exceed the dose levels which have produced significant statistical correlations of many health adverse effects.

#### 10. THE STANDARDS SETTING PROCESSES

#### 10.1 Setting exposure standards:

There is a strong contrast in the way in which western countries, led by the United States, and eastern countries, led by the Soviet Union, have gone about setting their safety limits for occupational and general public exposure to microwave radiation.

#### 10.2 United States research and standards:

The extremely haphazard approach of setting exposure standards in the United States is well documented in Staneck et.al (1980). Concern was raised in the 1940's in response to morale problems during World War II by popular fears of the effects of radar. After the war the beneficial uses of microwaves in medical services became widespread. Cataracts in dogs eyes were found at the Mayo Clinic in 1948 due to high level exposure to microwaves and other researchers reported a possible link between between microwaves, cataracts and testicular degeneration in dogs.

Interest in the biological effects of microwaves was rekindled in 1953 by John McLaughlin, a medical consultant to Hughes Aircraft Corporation, who drew up and sent to the military are report which listed purpura hemorrhagica (internal bleeding), leukemia, cataracts, head aches, brain tumors, heart conditions and jaundice as possible effects.

The U.S. Navy, through Kenneth Cole, Director of the Naval Medical Research institute, in a conference called for the purpose, attempted to determine the amount of radiant energy the body could handle under normal conditions. This is the source and basis of the attempt to avoid thermal effects of microwaves.

Cole reported "if I haven't misplaced a decimal point" then 1 W/cm² [1 million  $\mu$ W/cm²) is a dangerous level since a 70 km man, having a surface area of 3000 cm² (Cole's figure) and absorbing about one-third of the radiation coming from the source, would absorb nearly as much energy as he can eliminate through normal body functions. The group agreed on a safety factor of 10, giving 0.1W/cm² [100,000  $\mu$ W/cm²] as a reasonable dividing line between safe and hazardous exposures. Despite dissent from several members who sought a lower guideline, this was adopted.

A biophysicist who was at the meeting quickly discovered that the human body dissipates about 100 W, not the 150 W assumed, and that its area is close to 20,000 cm<sup>2</sup> not the 3,000 cm<sup>2</sup> assumed. Putting these figures together he estimated that the normal heat loss was 0.005 W/cm<sup>2</sup> so that 0.1 W/cm<sup>2</sup> was 20 time the amount the body could dissipate. 0.1 W/cm<sup>2</sup> was quickly abandoned and replaced by 10 mW/cm<sup>2</sup> [10,000  $\mu$ W/cm<sup>2</sup>] in official communications. These became the actual standard a decade later as C95.1.

The 1950's and 1960's were spent in debates and committees largely involving the military, communications and radar industry and the American Standards Association. It led to the continued return to 10 mW/cm² and the formal adoption of this as C95.1. C95.1 was always intended to be the best approximation for microwave exposure in the occupational group, NOT the general public. It was based on the clinical studies, personnel surveys, animal experiments and specific research of anomalistic effects. At the time it was adopted there was insufficient evidence and little motivation to soundly research, identify and verify the long-term effects of repeated moderate or continuous low levels of exposure.

The most obvious group to study was radar operators. A 1945 survey became the "standard". It concluded that there was "no evidence" of abnormalities. The survey was extremely superficial and very short term. Radar only came into use in the Second World War. They carried out not urinalysis nor blood chemistry analysis. However, there conclusion of "no significant changes" was not justified on the basis of the data they presented which showed a statistically significant increase in red blood cells in exposed workers and a high incidence of headaches.

Animal experiments, using quite high levels of relatively short term exposure, showed clear thermal effect but little in the way of conclusive, non-thermal effects. By 31 August 1967 the Tri-Service Programme concluded "the biological effects involves were (i) thermal, (ii) noncumulative, and (iii) of little concern since man has a built-in alarm system coupled to his threshold of pain that protects him from thermal injury.

The problems of basing this on short-term, high exposure experiments and yet workers in the military and industrial situations were exposed to long-term, low-level exposure. Papers were presented and published in the 1960's, pointing to defects in the procedures. Even at this time researchers were reporting reliable results of testicular damage in rats at 5 to 10 mW/cm², brain changes between 12 and 64 mW/cm², and changes in blood counts at about 13 mW/cm². More and more of the researchers were becoming open to the possibility of non-thermal effects at or below C95.1 . R.L. Carpenter brought a group together to gather the strands of research which pointed to non-thermal effects. Carpenter et.al (1960) established hazard thresholds well below 100 mW/cm²

Early in the 1950's, industrial corporations had adopted far stricter standards than the military, with Bell Telephone using a 100  $\mu$ W/cm² guideline as of 1953. On the other hand the military were still arguing against recognising non-thermal effects below 100,000  $\mu$ W/cm². The Carpenter group appeared to challenge the status quo. The question arises as to why was the standard C95.1 set at 10 mW/cm² "just when scientific research was beginning to reveal how much more work remained to be done"?

The simplest answer, and the one advanced by Paul Brodeur, a major critic of past policy, is that many people involved in setting C95.1 "felt obliged to protect the 10 mW level at all costs and to ignore, deny or, if the worst came to the worst, suppress any information about adverse effects of low-intensity microwave radiation". For Brodeur the values behind this are clear, "there was the belief in military preparedness and the presumption that a standard below 10 mW/cm² would interfere with national defense".

The truth may not be as simple as this but it is very clear that there was and still is a strong aversion to acknowledging even the possibility or adverse non-thermal effects in the official 'western' approach to standards setting which has reluctantly progressed downwards from high accepted exposure levels because the technology which produced high levels of exposure were 'necessary' for military security or industrial development. The failure of people to develop visible adverse health effects in the first few years was taken as 'proof' that there were, and would not be, adverse health effects in the future.

Recently the Institute of Electrical and Electronic Engineers (IEEE) proposed a 1 mW/cm² [1000  $\mu$ W/cm²] standard. Australia and New Zealand use a 1000  $\mu$ W/cm² occupational standard and a 200  $\mu$ W/cm² public exposure standard (NZS 6609). These are still based on a reluctant reduction, moving down from the thermal threshold rather than prudent avoidance on non-thermal effects which are now being suggested or identified at levels well below 200  $\mu$ W/cm². Despite the information contained in this report, and information allegedly contained in a C.S.I.R.O. in-depth review of the literature which identifies many non-thermal effects at exposure levels considerably below the 200  $\mu$ W/cm² standard (a report which is being withheld at present for suspicious reasons according to correspondence), there is now a proposal before a meeting of the joint Australia/New Zealand Standards Association subcommittee on radio frequency exposure standards, to raise the allowable exposure standard by a factor of FIVE.

#### 10.3 Bell Laboratories Internal Standards

Stenbeck et.al. report that in November 1953 "the Central Safety Committee of Bell Telephone had taken an even more conservative stand and adopted a 0.1 mW/cm [100  $\mu$ W/cm<sup>2</sup>] guideline based on a safety factor of 1000. A common public verses occupational safety margin is a factor or 10 which would place the comparative Public Safety level at 10  $\mu$ W/cm<sup>2</sup>. The reasons for this conservative approach are not given, but it should be reasonable for public authorities in New Zealand, with the legal framework of the RMA (1991), to set even stricter public exposure limits now, in the face of the growing epidemilogical evidence which is now available.

#### 10.4 Soviet Research and Standards:

In the Soviet Union, many experiments were carried out on animals and epidemiological studies were carried out relating to people. The WHO report "Environmental Health Criteria 16: Radiofrequency and microwaves" summarises this work. The author reports subjective complains of workers consisting of "headaches, irritability, sleep disturbance, weakness, decrease in sexual activity, pains in the chest and poorly defined feelings of ill health." He further notes that "On physical examination, tremor of the fingers with extended arms, acrocyanosis, hyperhydrosis, changes in demagraphism and hypotonia were reported in the USSR (Gordon, 1966)."

Gordon and her coileagues reported studies on occupationally exposed workers who were divided into 3 groups according to the levels of exposure to microwave radiation:

- (a) Periodic exposure at power densities from 100 to 10,000 μW/cm² (and higher) of maintenance personnel and workers, who had been employed in repair shoes since 1953;
- (b) Periodic exposure at power densities from 10 to 100 μW/cm<sup>2</sup> of technical maintenance workers, some users of microwave devices, and research workers, employed after 1060; and
- (c) Systematic low-level exposure of personnel using various microwave devices, mainly radar.

In the first two groups, including the group exposed to 1/20th to 1/2 of the New Zealand Standard NZS 6609, changes in the nervous and cardiovascular systems were reported. In the low exposure group similar symptoms were observed but they were less evident and easily reversed. About 1000 individuals were observed over a period of 10 years and some doubt exists about the exact exposure.

Given this kind of research result it is not surprising that the USSR adopted a 5  $\mu$ W/cm<sup>2</sup> standard for public exposure, to ensure that it avoided the evident problems of the middle and high occupational exposure groups.

The serious problems which still persist in determining actual exposure levels and actual health status of workers who work with microwaves, let alone the general public who are being exposed to gradually increasing background levels of microwave radiation in most cases, and in some cases, considerably greater than ambient levels when living or working in the vicinity of a cellsite.

#### 10.5 Conclusions about Standards setting:

Soviet research on actual and potential biological effects of microwaves has been very open to the possibilities of effects and many adverse effects have been identified and documented to a sufficient degree to set standards at very low levels using the Precautionary Principle of "Prudent Avoidance" if their is any possibility of adverse health effects.

In contrast to this, the United States work was based on the need to prove an adverse effect before a stricter standard could be adopted. It commenced with a conviction that proven effects include burns and body heating at high levels of exposure to microwaves. Burns and death have been observed when intensities of microwave radiation approach those of sunshine on a summers day, i.e.  $1000 \text{ W/m}^2$ ,  $100,000 \,\mu\text{W/cm}^2$ .

As a first objective the US standard was sought which would avoid these drastic effects, settling down to  $1000~\mu\text{W/cm}^2$ . This approach does not recognise, until reliably proven, any effects below this level. It is important to realise that the burning effect of the sun's radiation is caused almost entirely by the ultraviolet part of the spectrum, which has an intensity of about 100 to  $200~\mu\text{W/cm}^2$  near noon on a summers day, and, when weighted by the skin's response to UV is about  $29~\mu\text{W/cm}^2$ .

In the same way, some specific response of human bodies to microwave radiation is feasible, since many human body parts have a dimension about half of the microwave wavelength, making them a strong absorber like an aerial for a radio. The Soviet work recognises responses in human bodies at similar intensities to the weighted UV intensity and lower.

The military and industrial dominance of western standard setting has a clearly different approach to standard setting and the approach adopted in the Soviet countries. The mindset behind this dominates the official New Zealand position to this day and behind Telecommunication Industry proposals to raise the allowable exposure level by a factor of five, currently before the Standards Association Sub-Committee, stongly favours industry and military development freedom over public health protection.

In contrast, I strongly advocate that the Resource Management Act (1991), which requires the Avoiding, remedying or Mitigating of any potential or actual adverse effects on the environment (including people), means that were there exists a reasonable doubt about the safety of microwave exposure down to 10  $\mu$ W/cm² and lower, a public protection level should be set at 1/100th of this, i.e. 0.1 $\mu$ W/cm². This too should be an interim standard because of the indications which are already available, that children, pregnant women and human brains could well be adversely affected below this level.

There has always been a large communication gap between the technical and military community and the exposed public and workers. This gap has widened with time. Parallels with the nuclear power, chemical and pharmaceutical and the tobacco industries are strong and instructive. Recent class action litigation costing billions of dollars are real threats to industries who have contaminated large numbers of people, over many years, and finally there is sufficient evidence to convince a court. There have been decades of denial, followed by years of doubt and then finally there is sufficient weight of evidence for liability to be established and damages costs awarded.

This may or may not turn out to be the case for the industries which are progressively increasing the exposure of billions of people to low-level microwaves. But it cannot be ignored and must be a strong motivating factor in seeking to suppress any contrary debate.

However, suppressed debate only fuels fear and anxiety. Open debate with a frank sharing of information, resulting in prudent and conservative (low), publicly acceptable (not industry determined) exposure standards for the general public, would allay the fears and facilitate the use of the cleanest, most efficient technology.

#### 11. CONCLUSIONS:

Despite the limitations posed by the lack of monitoring of people's exposures to microwave and other radio-frequency electromagnetic radiation, and the current early stages of identifying particular biological mechanisms to account for the observed statistically significant relationships, the weight of evidence which is emerging is compelling.

It is physically possible and statistically probable that very low levels of microwave exposure, well below the current New Zealand exposure limit, significantly increase the chances of miscarriage for newly pregnant women, testicular cancers in men, behavioural changes and nervous dysfunction through resonant absorption of pulsed microwaves in people's brains, cardiovascular stresses, and a range of blood and genital cancers and biochemical changes. Breakdown of DNA by the molecular-resonant absorption of microwavs is the probable cause of cancers.

The search for mechanisms between the biological effects and doses of microwaves will continue. The opportunity for discovery will increase as exposure levels for the population at large continue to rise, provided that the appropriate monitoring and research is conceived, is funded and is carried out. In a special editorial comment (Editorial Commentary: epidemiology and exposure to electromagnetic fields, American Journal of Epidemiology, Vol 131, No.5, 1989) Richard Monson says in part:

"The challenge, therefore, is to conduct studies that measure current and future exposures in a precise and unbiased manner and to measure biologic outcome with precision, without error and in a timely manner." .... "The issues raised by the question of the possible health effects of exposure to electromagnetic radiation are prototypes for similar issues that will be faced by epidemiologists in the twenty-first century. Rather than conducting short-term studies that will "test a hypothesis", we must be developing systems to collect data on exposure and disease that will become part of the fabric of the community, the workplace and the healthcare organisation."

In the Resource Management Act legal environment it is sufficient that potential adverse effects on the environment have been identified. I believe that the epidemiological evidence shows actual significant adverse effects, but even the most ardent critic should be able to concede and accept the existence of potential adverse health effects.

"Avoiding, remedying or mitigating any adverse effect on the environment" (where effect is given the wide definition in Section 3 RMA (1991)) in the case of cellsites involves application of the precautionary principle by using the prudent avoidance of any exposure of the general population to avoid any actual or potential adverse health and behavioural effects. This can be accomplished within the parameters set by the evidence presented here by a spacing of cellsites at least 300 m from residences and places where people, especially children, remain for long periods.

The precedents of Planning Tribunal decisions require the fulfillment of section 5(2) (a), (b) and (c) of the RMA (1991) (protecting the environment) before considering the matters in section 5(2) (Human Development). This involves a total rejection of any tradeoff of adverse effects on the environment for one group of people against the economic gain of another. Hence any claimed economic dis-benefit of such a spacing requirement cannot be used to argue against the clear benefit of avoiding or significantly mitigating adverse effects on the environment.

#### Recommendations:

- a. Recognition of the legal position of the Resource Management Act (1991) requirement under the present High Court and Planning Tribunal precidents that the RMA gives a "greater emphasis on environmental protection" and the environmental "bottom line" of section 5(2) (a), (b) and (c) must be jointly fulfilled before consideration of the first part of section 5(2). When this is coupled with the section (3) definition of 'effect' means that there is a legal imperitive to "Avoid, remedy or mitigate any actual or potential adverse environmental effect which is cumulative over time or in combination with other effects regardless of the scale, intensity, duration or frequency of the effect, including any effect of potential effect of high probability or of low probability which has a high potential impact."
- b. Recognition of the epidemiolgical and biophysical evidence which strongly points to significant actual and potential adverse human health impacts due to the cumulative effects of continuous, pulsed, low levels of microwaves, such as those radiated from cellsites, with such adverse effects including brain function, DNA breakdown, Leukemia, miscarriage, testicular cancer, suicide, gential cancer and reduced immune system performance.
- c. Cellsite transmitters should not be located in or near schools, kindergartens, daycare centres or playcentres where children and pregnant mothers spend long periods of time. They should also be separated from schools and residences by such a distance that the intensity of the microwaves, when averaged over a year, does not exceed  $0.1~\mu\text{W}/\text{cm}^2$ .

- d. Wherever a cellsite is approved an adequate monitoring programme should be established, as recommended by Dr Richard Monson above. This would include systematic monitoring of exposure levels as well as appropriate health indicators in line with other Resource Consents to discharge contaminants under the RMA (1991).
- e. Active contact should be made and maintained with international work in progress on the effect of cellsite transmissions on populations with the results being used to guide policies, studies and standards in New Zealand.

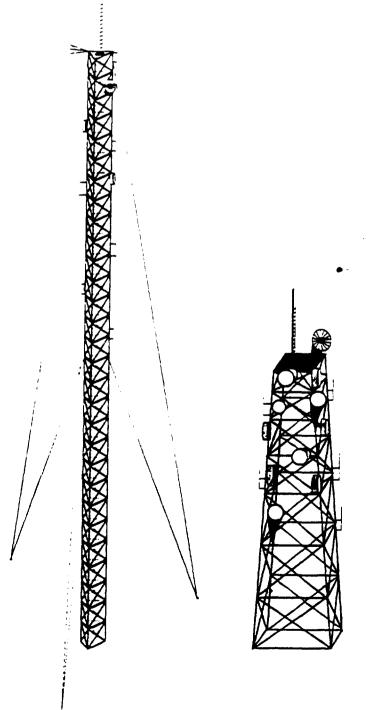
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# Summary and Results of the April 26-27, 1993 Radiofrequency Radiation Conference

Volume 1: Analysis of Panel Discussions





given the uncertainties that exist about the specific types of exposure to RF radiation incurred by human populations.

- Some uncertainty exists about whether RF radiation should continue to be considered a non-carcinogen. Some members of the panel felt that, because of this uncertainty. EPA should not continue to state with certainty that RF radiation is not a carcinogen.
  - (4) More research, including longer-term cohort studies that incorporate careful measurements of occupational exposure, including exposure type (pulsed vs. continuous wave, high vs. low frequencies, etc.), duration, and information about confounding variables (including multiple exposures) must be conducted before EPA takes further regulatory action to control human exposure to RF radiation.

Long term animal studies of radiofrequency and cancer association

The material enclosed shows:

1. Animal studies concerning cancer reported in the World Health Organization Environmental Health Criteria Report #137 on Electromagnetic Fields 300 Hz to 300 GHz, which includes mobile telecommunications frequencies.

The report shows on pages 149 and 150 a list which includes studies both above the 4 Watt per kilogram of body weight deemed safe for animals, and studies below this cut-off.

If we only consider studies at "safe" levels, below the 4 Watts of power per kilogram of body weight deemed safe and studies with long-term exposure (e.g. greater than 2 months), we find 4 studies

- o Szmigielski et al. 1982 showing increase in sarcoma colonies in the lungs due to implanted cells (level 2 to 3 W/kg)
- o Szmigielski et al. 1982 showing acceleration of mammary tumors in animals predisposed to such tumorss (level 2 to 3 W/kg)
- o Szmigielski et al. 1982 showing acceleration of skin tumors in animals painted with a carcinogen (level 2 to 3 W/kg)
  - o Guy et al. 1985 showing a more than 3 fold increase in spontaneous malignant primary tumors (level 0.4 W/kg)

There was also one study by Santini et al. which did not report effects but the longest lived animal only lived 690 hours after application of the carcinogen (less than 1 month), so the effect of microwaves may not have been able to take effect (animals were exposed 15 days to radiofrequency before application of the carcinogen)

Thus, the data show that at levels considered "safe" for animals, 100%, or all 4 out of 4 studies where animals lived more than 2 months after exposure show a positive association between cancer and radiofrequency exposure.

2. Statement from the EPA that "carcinogens pose a risk of health effects even at low levels of exposure...(and that) EPA calculates health risks estimates assuming that the risk of incurring either cancer or hereditary effects is linearly proportional to the dose received in the relevant tissue."

Therefore the above studies finding a positive association at exposure levels higher than would typically be experienced by the general population are still relevant to health risks at such lower exposure levels.

IF THIS AGENT WERE ANTHING OTHER THAN RADIOFREQUENCY FOR RADAR AND TELECOMMUNICATIONS THERE WOULD BE NO DOUBT THAT IT MAY PUBLICLY VEIWED AS A CARCINOGEN AND THAT THE PUBLIC EXPOSURE WOULD BE KEPT TO A MINIMUM.

GIVEN THAT SATELLITES MAY OFFER SUCH MINIMUM EXPOSURE, ON WHAT BASIS ARE WE ESTABLISHING A NATIONAL POLICY TO EXPOSE ARE CITIZENS TO LAND-BASED SYSTEMS, AND MOREOVER, FORCING LOCAL JURISDICTIONS TO ACCEPT SUCH FACILITIES CLOSE TO HOMES AND SCHOOLS?

#### THE ENVIRONMENTAL HEALTH CRITERIA SERIES

Acrolein (No. 127, 1991) Acrylamide (No. 49, 1985) Acrylonitrile (No. 28, 1983) Aldicarb (No. 121, 1991) Aldrin and dieldrin (No. 91, 1989) Allethrins (No. 87, 1989) Ammonia (No. 54, 1986) Arsenic (No. 18, 1981) Asbestos and other natural mineral fibres (No. 53, 1986) Barium (No. 107, 1990) Beryllium (No. 106, 1990) Biotoxins, aquatic (marine and freshwater) (No. 37, 1984) Butanols - four isomers (No. 65, 1987) Cadmium (No. 134, 1992) Cadmium - environmental aspects (No. 135, 1992) Camphechlor (No. 45, 1984) Carbamate pesticides: a general introduction (No 64, 1986) Carbon disulfide (No. 10, 1979) Carbon monoxide (No. 13, 1979) Carcinogens, summary report on the evaluation of short-term in vitro tests (No. 47. Carcinogens, summary report on the evaluation of short-term in vivo tests (No. 109. 1990) Chlordane (No. 34, 1984) Chlordecone (No. 43, 1984) Chlorine and hydrogen chloride (No. 2). 1982) Chlorobenzenes other than hexachlorobenzene (No. 128, 1991) Chlorofluorocarbons, fully halogenated (No. 113, 1990) Chlorofluorocarbons, partially halogenated (methane derivatives) (No. 126, 1991) Chlorophenois (No. 93, 1989) Chromium (No. 61, 1988) Cyhalothrin (No. 99, 1990) Cypermethrin (No. 82, 1989) 1,2-Dichloroethane (No. 62, 1987) 2,4-Dichlorophenoxyacetic acid {2,4-D} (No. 29, 1984) 2.4-Dichlorophenoxyacetic acid environmental aspects (No. 84, 1989)

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ALL 4 out of 4 long term **Environmental Health Criteria 137** Studies of Radiofrequency at "SAFE" doces show POSITIVE ASSOCIATION WITH CANCER **ELECTROMAGNETIC FIELDS** (300 Hz to 300 GHz)

· The AN, SI standard considers 4 watts/k:lopram at body safe for animals.

Published under the joint sponsorship of 08 watts Rilo the United Nations Environment Programme, the International Radiation Protection Association, and the World Health Organization



Let's see the findings of stuckes of long-lemme World Health Organization World Health Organization Geneva, 1993

Below 4 w/tg

<sup>.</sup> Out of print

frequencies (assessed as the decreased survival of implanted embryos and fetuses). Much experimental evidence suggests that acute or long-term RF exposures do not result in an increase in chromosome aberration frequency, when temperatures are maintained within physiological limits. One study reported an increased frequency of cytogenetic effects in mice exposed long-term at SARs between 0.05 and 20 W/kg. However, this study was not successfully corroborated using a different strain of mouse.

In general, the data in Table 28 suggest that the only exposures that are potentially mutagenic are those at high RF power densities, which result in substantial increase in temperature.

#### 7.3.10 Cancer-related studies

A summary of cancer-related animal studies is given in Table 29. The number and types of studies are limited.

Exposure to RF levels sufficiently high to induce hyperthermia has generally resulted in tumour regression following transplantation of tumour cells (Preskorn et al., 1978; Roszkowski et al., 1980). In contrast, an increase in tumour progression has been observed in mice exposed long-term at lower, possibly thermogenic, SARs (Szmigielski et al., 1982). This effect was related to a non-specific The authors suggested a transient shift in immune surveillance resulting in a lowering of resistance to neoplastic growth, as a likely explanation. Exposure at about 1 W/kg did not have any effect on melanoma growth in mice (Santini et al., 1988).

The effects of exposure on spontaneous or chemically-induced tumours have also been examined. In contrast to transplantation studies, these can test for an effect on the process of carcinogenesis. Two early studies (Prausnitz & Suskind, 1962; Skidmore & Baum, 1974), relevant to cancer induction, but in which the methodology was flawed in relation to an analysis of this end-point, are described for completeness. An increased incidence of monocytic leukosis (defined as a non-circulating neoplasm of white-blood cells) and lymphatic or myeloid leukaemia (defined as a circulating "leukosis") was reported in Swiss mice exposed to thermally significant levels (half the acute LD<sub>50</sub>) of 9.27 GHz pulsed RF, for 5 days per week

Table 29. Cancer related studies

Exposure conditions

Effect on exposed group

Reference

Transplanted tumour cells

Preskorn et al. (1978)

2.45 GHz (CW), 35 W/kg, for 20 min/day during days 11-14 of gestation; offspring injected with sercoma cells at 16 days of age exposed for 36 days

tumour incidence in sarcomainjected offspring of exposed pregnant mice; rectal temperature of dams rose over 2 °C; exposed nuce had increased longevity

Retarded turnour growth and

2.45 GHz (CW), 25 W/kg, 2 h/day for 7 days; Injection of varcoma cells in mice 14 days after, or just after. RF exposure

from 6 weeks of age to

12 months of stress

Temporary tumour regression followed by renewed turnour growth 12 days later, when exposure 14 days after tumour injection: accelerated tumour growth if exposed before implantation of tumour; lung metasteses increased

Roszkowski et al. (1980)

2.45 GHz (CW) 2-3 W/kg or 6-8 W/kg, 2 h/day, 107 6 days/week; nrice exposed

RF caused increase in Szmigielski et al. (1982) sarcoma colonies in lungs in nsice injected intravenously with these cells; chronic via confinement caused similar increase in lung turnours as

2-3 W/kg, but 6-8 W/kg produced

higher increase in tumours

surface area/animal, or in

exposed or control mice

No difference in mean tumour Santini at al (1988) mean survival time between

2.45 GHz (CW and pulsed) 10 W/m 1.2 W/kg prior to, and during. B16 melanome tumour transplantation and growth: exposed for 2.5 h/day, 6 times/week for 15 days, prior to injection of

2.45 GHz ICW, 2-3 W/kg

or 6-8 W/kg, 2 h/day, for

from 8 weeks of age to

12 months of stress

6 days/week, mice exposed

to same schedule until death which was less than

Spontaneous or chemically-induced tumours

Imonth

SAR dependent acceleration of Szmigielski mammary tuniours in risce

genetically predisposed to these tumours, and acceleration of skin tumours in mice painted with the carcinogen 3.4 benzopyrene (BP)

et al. (1982)



Low dose (below 4w/kg
AND

Long term 149 Cmore than 2 mon

148

Table 29 (continued)

Exposure conditions Effect on exposed group Reference 2.45 GHz (CW), 100 W/m<sup>2</sup> Increased development of Szmigrelski 🛆 chemically induced hepatomes at al. (1988) 4-5 W/kg, for 2 h/day, 5-6 days/week for a few months and sercomes in mice; survival of exposed mice decreased; This dose is increased frequency of skintumours in mice given subcarcinogenic dose of BP 2.45 GHz (10 pe pulses at Total incidence of neoplasta (1985) 800 Hz) square wavenot significantly different modulated at 8 Hz, from that in controls; however, 0.4 W/kg. continuous exposure Increased number of primery at 2:27 months of age malignancies (18) occurred early (life time Study of rats) in exposed group compared with controls (5)

for 59 days (Prausnitz & Susskind, 1962). However, the study suffered several deficiencies: leukosis and leukaemia were inadequately defined, infection may well have confounded the results, a large proportion of mice died without a cause of death being identified, and statistical analysis was absent (Roberts 1983; Kirk 1984).

Skidmore & Baum (1974) reported that exposure for 5 days per week for 33 weeks to very short pulses (5 ns rise time; 550 ms decay time) of high field strength (447 kV/m) pulsed at 5 Hz, resulted in a reduced incidence of leukaemia in AKR/J mice (which spontaneously develop a high incidence of lymphatic leukaemia between 26 and 52 weeks of age) compared with controls at the end of the exposure. However, the absence of a complete analysis of leukaemia incidence (and other causes of death) precludes any conclusion being drawn from this study. The authors also reported a zero incidence of mammary tumours in 1-year-old female Sprague-Dawley rats that had been exposed for 38 weeks; evaluation was probably premature for this end-point, the tumours occur spontaneously mainly in older rats. A later study (Baum et al., 1976) reported no effects on mammary tumour incidence and other lesions in rats exposed for 94 weeks.

Two studies merit particular attention. The long-term exposure of mice at SARs of between 2 and 8 W/kg resulted in an increase in

the number of sarcoma cell colonies in the lungs (following the injection of sarcoma cells), as shown in Fig. 22, and in an SAR-dependent increase in the rate of development of spontaneous mammary tumours and chemically-induced skin tumours. Repeated microwave exposure, followed by a "sub-carcinogenic" dose of carcinogen, resulted in an increased number of skin tumours. A study of 100 rats exposed for most of their lifetime at about 0.4 W/kg did not show any increased incidence of non-neoplastic lesions compared with control animals; longevity was very similar in both groups. However, the overall incidence of primary malignancy in the exposed group (18) was significantly greater than the control

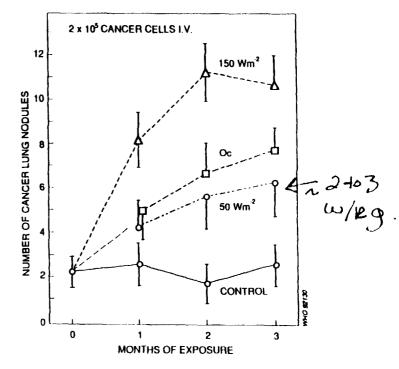


Fig. 22. Number of lung tumours (following intravenous injection of  $2\times10^5$  viable sarcoma cells) in mice exposed to 2.45 GHz microwaves or non-specific stress (overcrowding;  $O_c$ ). From Szmigielski et al. (1988).

THE THE WASHINGTHE THE STIDE

value (5), but was reported to be similar to the spontaneous incidence given in the literature for the particular strain of rat. Under these circumstances, it is difficult to draw any firm conclusions.

Tumour weights were not significantly different in rats implanted with mammary adenocarcinoma tissue and either exposed 25 days later to 2 kHz magnetic fields of up to 2 mT for 1 h a day for 9 days or not exposed (Baumann et al., 1989). Handling and restraint stress in animals were identified as possible confounders for the detection of subtle magnetic field effects.

#### 7.3.11 Summary and conclusions

Most of the biological effects of acute exposure to RF fields are consistent with responses to induced heating, resulting either in rises in tissue or body temperature of about 1 °C or more, or in responses for minimizing the total heat load. Most responses in different animal species, exposed under various environmental conditions, have been reported at SARs above about 1.2 W/kg.

These animal (particularly primate) data indicate the types of response that are likely to occur in humans subject to a sufficient heat load. However, direct quantitative extrapolation to humans is difficult, given species differences in responses, in general, and in thermoregulatory ability particularly.

The most sensitive animal responses to heat loads are thermoregulatory adjustments, such as reduced metabolic heat production and vasodilation, with thresholds ranging between about 0.05 and 5 W/kg, depending on environmental conditions. However, these reactions form part of the natural repertoire of thermoregulatory responses that serve to maintain normal body temperatures.

Transient effects seen in exposed animals that are consistent with responses to increases in body temperature of 1 °C or more (and/or SARs in excess of about 2 W/kg in primates and rats) include the reduced performance of learned tasks and increased plasma corticosteroid levels. Other heat-related effects include temporary haematopoietic and immune responses, possibly in conjunction with elevated corticosteroid levels. The most consistent effects observed are reduced levels of circulating lymphocytes and increased levels of

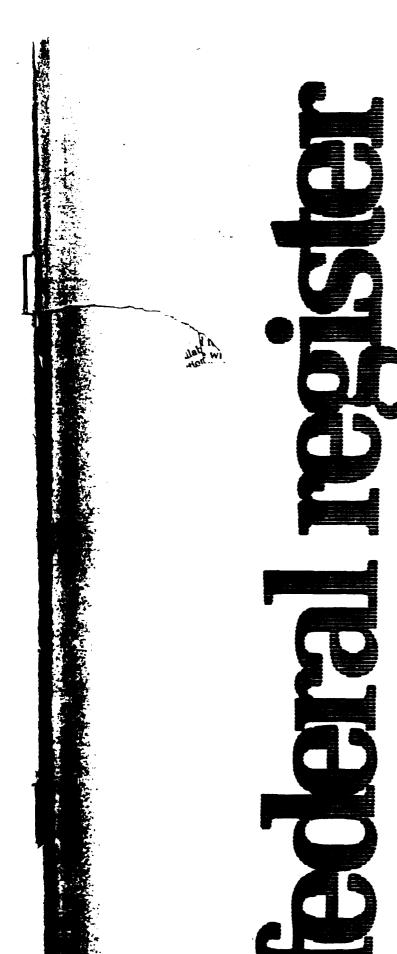
neutrophils, decreased natural killer cell function, and increased macrophage activation; an increase in the primary antibody response of B-lymphocytes has also been reported. Cardiovascular changes consonant with increased heat load, such as increased heart rate and cardiac output, have been observed, together with a reduction in the effects of drugs, such as barbiturates, the action of which can be altered by changes in circulation and clearance rates.

Most animal data indicate that implantation and the development of the embryo and fetus are unlikely to be affected by exposures that increase maternal body temperature by less than 1 °C. Above these temperatures, adverse effects, such as losses in implantation, growth retardation, and post-natal changes in behaviour, may occur, with more severe effects occurring at higher maternal temperatures.

Most animal data suggest that low RF exposure that does not raise body temperatures above the normal physiological range is not mutagenic; thus, such exposure will not result in somatic mutation or hereditary effects.

There is much less information describing the effects of long-term, low-level exposure. So far, it is not apparent that any long-term adverse effects can result from exposures below thermally significant levels. The animal data indicate that male fertility is unlikely to be affected by long-term exposure at levels insufficient to raise body and testis temperatures. Cataracts have not been induced in rabbits exposed at 100 W/m<sup>2</sup> for 6 months, or in primates exposed at 1.5 kW/m<sup>2</sup> for 3 months.

A study of 100 rats, exposed for most of their lifetime at about 0.4 W/kg, did not show an increased incidence of non-neoplastic lesions or total neoplasias compared with control animals; longevity was very similar in both groups. There were differences in the overall incidence of primary matignancies, but these could not necessarily be attributed to the RF exposure. The possibility that exposure to RF might influence the process of carcinogenesis is of particular concern. So far, there is no definite evidence that RF exposure does have an effect, but there is clearly a need for further studies to be carried out. Overwhelmingly, the experimental data indicate that RF fields are not mutagenic, and so they are unlikely to act as initiators of carcinogenesis. In a few studies, evidence has been sought of an enhancement of the effect of a known carcinogen.



Friday December 15, 1989

Part II

## **Environmental Protection Agency**

40 CFR Part 61

National Emission Standards for Hazardous Air Pollutants; Radionuclides; Final Rule and Notice of Reconsideration people receive as a result of these emissions is typically lower than their natural background dose, the resulting risk can still be significant. A source does not present an acceptable risk simply by being less than natural background. It is important to note that total background radiation from all sources, including naturally occurring radon, results in a calculated individual lifetime risk of fatal cancer of approximately one in one hundred. In most cases, little can be done to reduce most of this radiation exposure which people receive from natural background.

Industrial sources of radionuclide emissions in the air include a wide variety of facilities, ranging from nuclear power facilities to hospitals to uranium mill tailing piles. Industry uses hundreds of different radionuclides in solid, liquid. and gaseous forms, emitting different types of radiation (alpha, beta, gamma) at various energy levels. Industrial sources of radionuclide emissions fall into two major categories. The first include industries that use radioactive materials and have emissions as a result of an inability to completely contain the materials they use. For example. hospitals use radionuclides as part of their radiology departments. Since many of the radionuclides they use are gases. liquids capable of evaporation, or solidcapable of sublimation, some radionuclides inevitably are released into the environment. The other type of source is that which releases radionuclides (usually radon) as an unintended consequence of another activity, such as mining or milling. An example of this is phosphogypsum stacks (piles). These piles of waste material emit radon because radium (from which radon is produced by radioactive decay) is found naturally in the same soils that are the source of phosphate rock.

#### B. Health Effects of Radiation

The level and type of hazard posed by radionuclides vary, depending on such characteristics as the radionuclide's radioactive half-life, the type of radiation it emits, the energy level of the emission(s), and its ability to concentrate in the body. Different radionuclides will irradiate different parts of the body causing different types of cancers.

There are three major types of longterm health impacts from exposure to radiation: Cancer, hereditary effects, and developmental effects on fetuses such as mental retardation. Since there is such a strong foundation for quantifying the risk of fatal cancer, EPA's consideration of fatal cancers is the principa' health consideration in this

rulemaking. However, it is important to note that other health effects have also been considered in the rulemaking. The other effects are not specifically addressed in this discussion because none of them pose a more severe risk to health. In addition, risk distribution of health effects from radiation from most of the sources considered for regulation show that fatal cancers occur much more frequently than non-fatal cancers and cancers generally occur more often than genetic or developmental effects. For sources that emit radon, no genetic or developmental effects, and very few non-fatal cancers are expected.

Numerous studies have demonstrated that radiation is a carcinogen. It is assumed that there is no completely risk-free level of exposure to radiation to cause cancer. Health effects from radiation have been observed in studies of occupationally exposed workers and of the survivors of the Hiroshima and Nagasaki atomic bombs. This information has been verified with studies of animals in laboratories. However, the effects of radiation doses at low levels of exposure can only be predicted by extrapolating from the observed effects at higher doses since we do not have direct evidence of cancer causation at low exposure levels. Some pollutants cause diseases that are unique to the pollutant; for example, asbestos causes asbestosis. Radiation, however, causes some of the same types of cancers, e.g. leukemia and lung and liver cancer, that are caused by other factors. Since these cancers are not uniquely associated with radiation, it is not possible to differentiate cancers caused by radiation from other cancers.

The second type of effect is the induction of hereditary effects in descendants of exposed persons, which vary in degree and effect and may even be fatal. It is assumed that there is no completely risk-free level of exposure for hereditary effects. Although hereditary effects have been observed in experimental animals at high doses, they have not been confirmed at low doses in studies of humans.

Based on extensive scientific evidence. EPA believes it prudent to assume that carcinogens, including radionuclides, pose a risk of health effects even at low levels of exposure. Based on this science policy judgment. EPA calculates health risk estimates assuming that the risk of incurring either cancer or hereditary effects is linearly proportional to the dose received in the relevant tissue. However, the severity of either effect is not related to the amount of dose received. That is, once a cancer or an hereditary effect has been

induced, its severity is independent of the dose.

Regarding cancer, there continues to be divided opinion on how o interpolate between the absence of radiation effect at zero dose and the observed effects of radiation (mostly at high doses) in order to estimate the most probable effects at doses that represent small increases above natural background radiation. Most scientists believe that available data best support use of a linear modelfor estimating such effects. Others, however, believe that other models, which usually predict somewhat lower risk, provide better estimates. These differences of opinion have not been resolved to date by studies of the effects of radiation in humans, the most important of which are those of the survivors of the Hiroshima and Nagasaki atomic bombs.

Some studies have recently been completed, and others are now underway to reassess radiation dose calculations for the survivors of the Hiroshima and Nagasaki atomic bombs and to provide improved estimates of risk. These studies may reduce the uncertainty associated with extrapolation from high doses to low doses. These studies may also result in an increase of the estimated risk per unit dose. But they will not address the question of whether a threshold exists. EPA is monitoring the progress of this work and will initiate reviews of the risks of exposure to low levels of radiation upon its completion.

#### C. Risk Assessment

### 1. Risk Measures Considered in NESHAP Policy

In decisions on cancer risks from stationary sources of hazardous air pollutants, the Agency has estimated three measures of health risk. These are termed "maximum individual risk", "risk distribution", and "incidence". Each of these combines an estimate of the dose/response for a pollutant with estimates of exposure to the pollutant. The response estimated is the pollutant-related increase in the probability that an individual will contract fatal cancer in his or her lifetime. The exposure estimated is the average daily exposure assuming exposure for 70 years.

a. Maximum Individual Risk. Individual risk is expressed as an estimated probability, e.g., 1 in 100 ( $10^{-2}$ ), 1 in 1.000 ( $10^{-3}$ ), 1 in 10,000 ( $10^{-4}$ ). Thus, a  $1\times10^{-3}$  individual risk is an added "chance" of 1 in 1.000 of contracting fatal cancer sometime in the individual's lifetime.